

## Early Journal Content on JSTOR, Free to Anyone in the World

This article is one of nearly 500,000 scholarly works digitized and made freely available to everyone in the world by JSTOR.

Known as the Early Journal Content, this set of works include research articles, news, letters, and other writings published in more than 200 of the oldest leading academic journals. The works date from the mid-seventeenth to the early twentieth centuries.

We encourage people to read and share the Early Journal Content openly and to tell others that this resource exists. People may post this content online or redistribute in any way for non-commercial purposes.

Read more about Early Journal Content at <a href="http://about.jstor.org/participate-jstor/individuals/early-journal-content">http://about.jstor.org/participate-jstor/individuals/early-journal-content</a>.

JSTOR is a digital library of academic journals, books, and primary source objects. JSTOR helps people discover, use, and build upon a wide range of content through a powerful research and teaching platform, and preserves this content for future generations. JSTOR is part of ITHAKA, a not-for-profit organization that also includes Ithaka S+R and Portico. For more information about JSTOR, please contact support@jstor.org.

## PSYCHOLOGICAL LITERATURE.

## I.— NERVOUS SYSTEM.

Ueber Faserschwund in der Kleinhirnrinde. ADOLPH MEYER. Archiv f. Psych. u. Nervenkrankheiten. Bd. XXI, H. 1; mit 1. Taf.

The network of fibers in the granular layer of the cerebellar cortex was the portion examined by the author. The loss of fibers here was found to take place to a varying extent, and for convenience he distinguishes those cases in which it is slight, medium, and excessive; specimens were stained with Weigert's Hæmatoxylin. He then cites fifteen cases which he has himself examined. Dividing these according to the degree of degeneration of the cerebellar fibers into three groups, they form: Group I, Case 1. General progressive paralysis; in which degeneration was slight.—Group II, Cases 2-6 inclusive. Three of these were general progressive paralysis, one melancholia with stupor, one chronic paranoia. Degeneration medium.—Group III, Cases 7-14 inclusive. Seven of these were general progressive paralysis, one dementia senilis. Degeneration excessive.

The 15th case (idiocy) falls outside of any of these groups, being most probably a case of arrested development. The prominent characteristic in these cases was dementia. There was always a loss of fibers in the cerebral cortex—and the loss in the cerebella cortex appeared to follow on that in the cerebrum, and to be a slow process. The cause is entirely obscure, but the course of the fibers involved is taken to be through the middle peduncle of the cerebrum to the pons, and so to the cerebrum. The author looks forward to pursuing the investigation more in detail.

The author looks forward to pursuing the investigation more in detail. (The apparently close relation thus developed between the cerebral and cerebellar cortex, and association of degeneration of the fibers with dementia in this general way, are both facts of great value.—Rev.)

Recherches sur la localization des conducteurs des impressions sensitives dans les diverses parties de l'encéphale et sur la pathogenie de anesthesies de cause encéphalique. M. Brown-Sequard. Archives de physiologie, etc., No. 3, Juillet, 1889.

The author opens with the following propositions:

1. Each half of the brain is able to perceive sensory impressions arising in the two halves of the body. 2. The sensory elements are so distributed in the brain that sensation remains even when a large portion of the two halves of the brain has been destroyed. 3. The transmission of sensations still occurs even when the cord has been completely severed by two hemisections at different levels and on opposite sides, provided they are sufficiently distant from one another. 4. If we attribute the anaesthesia following organic lesions of the brain or cord to loss of function in the part injured, we are compelled to admit absurdities. . . . . As a matter of fact clinical experience shows that anaesthesia may or may not appear whatever the location of the organic lesion. 5. Anaesthesia, due to brain lesions, may occur on either or both sides when the lesion is single, or on one side when the brain lesion is double, or may disappear while the brain lesion at the same time becomes more extensive. 6. In the case of partial organic brain lesion the anaesthesia is therefore not due to the loss of function in the

nervous elements destroyed, but to an action of the lesion on the nervous matter about it causing inhibition of the sensory apparatus. This is a dynamic process, hence subject to great variation, thus giving

rise, under different conditions, to very various results.

As evidence for the above from the experimental side, he presents the results obtained from dogs in which lesion of the internal capsule, lateral portion of the base of the brain, or superior part of the cervical cord was followed by hyperaesthesia of the corresponding, anaesthesia of the opposite side. If, now, a hemisection of the cord be made on the side opposite to the initial lesion (at the level of the last dorsal or first lumbar vertebra) the anaesthesia and hyperaesthesia change places. In these experiments anaesthesia is most complete after section of the internal capsule, and diminishes according to the parts operated, in the following order: pons and lumbar cord; cerebral peduncle and cervical cord; medulla. Passing to the clinical data he divides his material (1) into cases with direct anaesthesia and (2) those with both direct and crossed anaesthesia, due to a lesion of one side only. For (1) he gives 59 cases, and for (2) some references to the literature of the subject. He adds that several investigators have found that anaesthesia of cerebral origin disappears on faradization of the skin.

The clinical evidence presented for this view is certainly open to the objection of not being critically collated. Supposing the experimental facts to be correct, the mere statement that the phenomenon is one of inhibition amounts simply to the statement that something does not

occur, and as it stands is no explanation at all.

Der Hund ohne Grosshirn. Prof. GOLTZ. XIV Wanderversammlung südwestdeutscher Neurologen und Irrenärzte, Mai, 1889. Original Bericht von Dr. L. Laquer.

Goltz communicated his observations on a dog which had lived 51 days after the removal of his fore-brain. The fore-brain on both sides was removed together with corpora striata, leaving only a small remnant about the brain-axis between the optic tracts. The thalami were of course secondarily involved. The remaining portions of the stem were soft and but poorly sculptured. The important point was that the dog lived so long a time after such an injury, and could, moreover, stand, walk and rise on his hind legs. He could not eat or drink alone but could chew food put well back in his mouth. Waking and sleeping alternated with him as with a normal animal. When hungry he was restless, when satisfied he slept. He could be waked by touching him at any point of the skin. He then opened his eyes, previously closed, and stretched like a normal animal on waking. If the limbs were put in an uncomfortable position he moved back to the normal. As occasion demanded he could whine, growl, bark and howl. Evacuating faeces or urine he took the positions of a normal dog. To sound he did not react. The senses of smell and sight were wanting because the nerves were sectioned.

Ueber das Rindencentrum für die Stimmbildung. ROSSBACH. Jahressitzung des Vereins der deutschen Irrenärzte. Jena, Juni, 1889. Abstracts of communications in Neurolog. Centralbl., No. 13, 1889, by Bruns.

The patient had symptoms of compression in the caudal cervical region, which at autopsy were found as due to a tumor. Further there was on the left side paralysis of the facial, atrophy of the tongue and paralysis of the vocal cord, of ten years standing. The autopsy showed a so-called encephalitis subcorticalis of the right inferior parietal lobe, of the posterior central convolution, where it helps to form the operculum, and of the posterior convolution of the island of Reil. In the medulla the nucleus of the hypoglossus was alone atrophied, whereas the nuclei